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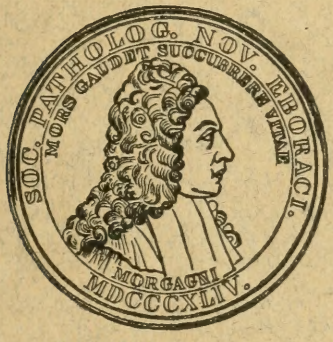
NEW YORK PATHOLOGICAL SOCIETY



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Proceedings
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Dr. James Ewing, *President.*

THE SIGNIFICANCE OF INTRAMEDULLARY DEGENERATIONS.

M. G. SCHLAPP, M.D.

At a recent meeting of this Society, I presented sections through the spinal cord, medulla, pons, and through the region of the neucleus of the third nerve, in a case of tumor of the right hemisphere of the cerebellum. These sections were presented in order to show degenerations in the posterior columns (which have been described by other writers), and similar degenerations in the intramedullary fibres of the sensory cranial nerves which, to my knowledge, have not been described before.

The specimens which I am showing you to-night are in addition to the aforesaid sections of the cerebellar case, transverse sections of the spinal cord of 1st.—A case of crushed cord. In this case the spinal cord was crushed in the region between the 6th and 7th dorsal vertebræ, absolutely destroying the cord in this region. If you will examine the sections of this case, which were taken from the lumbar and sacral regions, you will see that the posterior root-zone of Flechsig shows a marked degeneration. This degeneration can be followed to a point where the posterior root leaves the spinal cord, sometimes a little beyond; and if you will look closely you will see that this degeneration always stops, with the exception of a very few fibres, where the sheath of Schwann begins. These degenerated fibres are all ascending fibres and short ones, because they enter the gray matter where they seem to end.

2nd.—A case of transverse myelitis in the cervical region. In this case as in the crushed cord case, the posterior root-zone was markedly degenerated in the dorsal, lumbar and sacral regions.

3rd.—A case of alcoholic polyneuritis in combination with mental disturbances (described by Korsakow). In this case the degeneration was also well-marked in the posterior root-zone. The degeneration, however, was also found in other parts of the spinal cord, which I shall report later.

In all these four cases:

Cerebellar tumor case;

Crushed cord case. Lesion in the middle dorsal region;

Transverse myelitis case. Lesion in cervical region;

Alcoholic polyneuritis;

a marked lesion was found in the posterior root-zone. In the alcoholic case the nerves were involved; in the cases of transverse lesions of the cord, the descending tracts were all involved below the lesion; in the brain tumor case, the degeneration was almost entirely confined to the columns of Burdach, and especially in the posterior root-zone, a few degenerated fibres in the columns of Goll. The tendon and skin reflexes were missing below the lesion in the cases of transverse lesion of the cord. In the alcoholic case they were missing in the legs and lower part of the body, in the arms they were very much diminished, sometimes not obtainable, shortly before death missing. In the brain tumor case, they were entirely missing a few weeks before death.

Much has been written about the condition of the reflexes in cases of transverse lesion of the spinal cord. Bastian was the first, I think, to call attention to the absolute loss of the tendon reflexes of the legs in cases of transverse lesion of the cord in the cervical or upper dorsal regions. His explanation of the loss of the reflexes in the above conditions is that in a complete lesion of the cord the fibres which are constantly transmitting a stimulus from the cerebellum to the motor cells in the anterior horns of the spinal cord, whereby the muscles are kept in a normal tonic condition, are cut and this constant stimulus cannot reach the motor cells lying below the lesion, hence the muscles connected with these cells are not in a normal tonic state. Therefore, the reflexes which depend upon this tonic state are lost in the muscles involved. Recently cases have been described in which there were absolute transverse lesions of the cord in the cervical and dorsal regions with reflexes present or exaggerated below the lesion. The case of Kausch is especially important in demonstrating this fact. So the hypothesis of

Bastian cannot be accepted. The degeneration of the nerve fibres in the posterior root-zone of Flechsig no doubt explains the absence of the reflexes in these cases. In the alcoholic polyneuritis, the changes in the nerves could in themselves be sufficient to cause the loss of the reflexes, but the degenerations in the nerves are not so marked as the degenerations in the posterior root-zone of Flechsig.

It seems that the short fibres entering the cord through the posterior roots, or reflex fibres, are the ones in which the degeneration first takes place. Or in other words, they cannot withstand the effects of toxic substances or imperfect circulation as well as the fibres that carry the sensory impressions to the higher centres. This is also supported by clinical observations; that is, the absence of the reflexes is usually the first symptom of a disease in which the posterior columns are affected:—Tabes Dorsalis, ergot poisoning, brain tumor, etc. If the cause producing these degenerations continues long enough then of course the fibres transmitting the sensory impressions to higher centers will also degenerate, and we will then have a disturbance of sensation.

These cases will be published in full very soon.

A LYMPHOCYTOZOON OF THE GUINEA-PIG.

L. B. GOLDHORN, M.D.

While examining the blood of a certain guinea-pig, I found in the cytoplasm of a large number of lymphocytes highly refractive, round or elipsoid, non-motile bodies which when stained with ordinary methylene blue looked like vacuoles. As the nucleus of these cells was considerably displaced and undergoing karyorrhexis ac-

cording to the size of the vacuoles, it was suspected that a parasite was present. Upon staining smears with polychrome methylene blue a brilliantly stained body was seen, varying in size from a barely visible dot to more than the size of the nucleus of the lymphocyte. While it frequently filled the space in the lymphocyte completely, it would as frequently appear surrounded by a sharply defined achromatic zone. This latter appearance was rather constant with the small forms.

It was assumed that if this body were a parasite it might be transmitted by inoculation. Intravenous injection was made into one young guinea-pig, while a second one received an equal amount of blood intraperitoneally; and into a third one a subcutaneous injection was made. One control pig was kept with these young ones, and all were isolated from the old infected pig. The transfused blood was suspended in physiological salt solution. All the animals were first carefully examined for abnormalities in the lymphocytes with negative results. It was thought best to take young animals since old animals appeared to show the bodies above described in nearly half the number examined.

At first, blood examinations of the inoculated pigs were made daily with entirely negative results; at the expiration of three weeks very diminutive bodies surrounded by a clear space were seen in the cytoplasm of the lymphocytes of the pig in which the injection had been made intravenously. The nucleus of such lymphocytes did not show any degenerative change. Three weeks later the the parasites had increased to twice their first size, but numerically they were not increased. At the very best few could be found.

The animal on which these observations were made showed as many infected lymphocytes as normal ones.

It was a pregnant female. The two young ones did not show at birth (nor do they show to this day) lymphocytes containing parasitic bodies.

Lymphocytes frequently showed more than one parasite. The only way of telling the small forms from the metachromatically staining bodies so frequently seen in the cytoplasm of lymphocytes was by observing the clear space surrounding the parasitic bodies. In multiple infection of one cell one parasite was not infrequeutly observed to be larger than others in the same cell. Two parasites maturing side by side were seen from time to time. I did not succeed in demonstrating anything in the clear space surrounding the very small forms; but this was very different with the large ones. In the first place it was noticed that the latter frequently have a more compact central body. The largest forms consisted of a loosened chromatin body surrounded by a meshwork which occupied frequently the entire achromatic zone mentioned. I have come to the conclusion that this clear zone is at all times made up of a substance the nature of which is still undetermined. I succeeded in finding a couple of extra cellular bodies surrounded by a sharply defined envelop corresponding in width to the achromatic area of half grown intra-cellular forms. In other instances lymphocytes injured in spreading the blood were found to have discharged their parasite which was found lying free in plasma, surrounded by a meshwork.

It is probable that Kurloff's cells with vacuoles are those described in this paper, and therefore I give in full the quotation by Ehrlich, since the original by Kurloff is in Russian. He says:

"This is a quite peculiar group, characteristic for the blood of the guinea-pig. It shows transitions in the blood from large mononuclear to transitional and poly-

nuclear forms, but is marked by the lack of any kind of granulation. Instead of the latter we find in these cells a roundish, nucleus-like form in the protoplasm, which also takes the nuclear stains, and possibly is to be considered an accessory nucleus. We have received the impression that we have here to deal with a vacuole filled with substance secreted by the cell. In a large series of preparations, it is possible to obtain some elucidation of the development and fate of these appearances. They first appear in point-like granules in the protoplasm, bearing no relation to the cell-nucleus; they gradually increase and acquire a considerable circumference. When they have attained about the size of the cell-nucleus, they, or rather their contents, appear to break through the protoplasmic membrane and to leave the cell. The number of the vacuoles containing cells is 15 to 20% of the colorless blood corpuscles."

Cells with wide meshes in their cytoplasm simulating vacuoles are not infrequently seen in smears, but they are altogether unlike the bodies in question. To regard this body as an accessory nucleus is out of the question. It is seen at times outside of the cell as already stated, and is then frequently sharply outlined by a more or less achromatic zone. Moreover, its affinity for stains is altogether unlike that of the nucleus of the lymphocytes, and it stains best metachromatically.

The statement that vacuoles containing cells of the normal guinea-pig number 15 to 20% of the colorless corpuscles is an error if Kurloff's vacuoles are identical with the bodies described because there are many guinea pigs, if not the majority of them, that are free from such bodies.

If they are really parasites it may be desirable to examine the blood of guinea-pigs which are to be used

for serum reactions to determine if they are heavily infected, as they then might occasionally alter such reactions.

I shall be thankful for suggestions as to the nature of these bodies. A microscopic examination of the tissues from guinea-pigs whose lymphocytes contained these bodies showed that there were in the alveoli of the thyroid gland what appeared to be foreign bodies, sometimes closely resembling encysted parasites; between the epithelial cells lining the alveoli, smaller foreign bodies were also seen. Future investigation will show how constant these changes are. The bone marrow was seen to contain the bodies in lymphocytes in large numbers. The technique was as follows:

Smear were fixed in wood alcohol for 15 to 30 seconds, and then washed in water until the water adheres to the smears. The preparations were stained in polychrome methylene blue (Goldhorn) for a few seconds, long enough to have the smears look grayish or bluish, and they were then washed thoroughly in clean water. Finally they were stained in .1% eosin for from 15 to 30 seconds, and washed again. Short eosin staining will leave the red corpuscles greenish, while longer eosin staining will give pinkish red corpuscles. The specimens were dried in air and mounted in balsam.

Discussion.

Dr. Ewing: I have seen the specimens now exhibited and have had an opportunity of examining them, but I am unable to say whether they are parasites or not, though from their appearance I am inclined to think they are.

PARATYPHOID INFECTIONS.

WARREN COLEMAN, M.D. AND B. H. BUXTON, M.D.

Gilbert proposed the terms "paratyphoid" and "paracolon" in 1893, to designate the intermediates of the typhoid-colon group of bacilli. Durham calls the intermediates the "Gaertner-group." The group includes Bacillus enteritidis, Bacillus psittacosis, Bacillus paratyphosus, Bacillus choleræ suis, and some others.

The intermediates are pathogenic for man; Bacillus enteritidis induces epidemics of meat poisoning, Bacillus psittacosis a peculiar epidemic broncho-pneumonia with typhoid symptoms, and Bacillus paratyphosus a disease closely similar to typhoid fever.

Attention is confined in our paper to paratyphoid fever. Thirteen cases have been reported in whose blood, stools, urine, or complicating inflammatory processes paratyphoid bacilli have been found. Four cases have been reported in which the diagnosis was made by the serum reactions. We report a fourteenth case, clinically identical with typhoid fever, in blood of which a paratyphoid bacillus was found.

An analysis of the symptoms of the reported cases shows that the disease closely resembles typhoid fever. All the cases were regarded as such until a bacteriological examination revealed their true nature. None of the cases gave the Widal reaction, and it is thought that other cases of clinical typhoid fever which have failed to give the reaction may have been paratyphoid infections.

Bacteriologically, the bacillus isolated from the case closely resembled Bacillus typhosus in many biological characters, but produced gas in glucose media.

This case shall be reported in full later.

Discussion.

Dr. N. E. Brill: I would like to say a few words in reference to the clinical aspect of the subject. About five years ago I called the attention of the medical profession to a group of cases which clinically bore some resemblance to typhoid fever, but in which the Widal reaction remained persistently absent throughout the disease and subsequent convalescence. There could be no doubt about the absence of the Widal reaction as examinations were made every day in each case, not only during the stay in the hospital, but after they had left. At that time I was impressed with the specifity of the Widal reaction, and on that account I attempted so far as my resources permitted and the facilities of the laboratory at Mount Sinai Hospital allowed, to determine the cause of the infection in the seventeen cases of this class which came under my observation. It should be remembered that at that period, outside of Elsner's and Hiss' media, no differential method of culturing the Eberth bacillus had been suggested, nor had any attempts been made to differentiate the organisms whose biological situation was intermediate between the typhoid bacillus at the one side and the colon bacillus at the other. The bacteriological work, therefore, while it was serious, could not be taken when viewed in the light of present methods as very reliable. It was sufficient, however, to prove that in the feces and in the blood withdrawn from the spleen —a procedure which I would not now countenance—of the cases reported the typhoid bacillus was absent. A bacillus answering to the ordinary tests of the colon bacillus was isolated. At that time, bearing in mind the specific nature of the Widal test, we tried the reaction of the blood in each of the seventeen cases with a culture of the isolated bacillus, but failed to get a reaction. In

two of the cases the effect of the blood of the patients upon a culture of the bacillus obtained from each patient respectively failed to give a reaction. This shows that at the time I appreciated the fact that some other organism than the one isolated was the offending and disease-producing organism. The bacteriological work was finished before I read of Widal's observation of a paracolon bacillus from an œsophageal abscess which had casually come into the hospital in Paris. There was no clinical observation in the case of Widal's, as I remember it.

When I presented my paper based on the examination of these seventeen cases I insisted upon the view that these patients did not suffer from typhoid fever, but from some other infection. One of the strongest factors which induced me to form this belief was the fact that a nurse at the training school at the hospital had had an undoubted case of typhoid, six months before, with a Widal reaction, and became ill again with an infection which clinically was similar to the first but in which the Widal reaction was persistently absent, and in blood aspirated from the spleen no typhoid bacilli could be found, though there was found a bacillus which I at that time regarded as the ordinary colon bacillus.

My view of the presence of another than the typhoid infection was bitterly assailed, and in speaking of the cases with Dr. Osler, he regarded them as typhoid. Shortly afterwards, Norman B. Gwyn of Johns Hopkins, published a case which was similar to those I had previously reported and from whose blood he had isolated a paracolon bacillus. From that time on (from 1898) I insisted at various societies that my cases were not cases that should be classified under the head of typhoid fever, but that they were most probably the result of a disease due to a paracolon infection. The expression of this

view has always called forth opposition, and it was only three months ago that the worthy President of this Society, Dr. Ewing, fiercely assailed it on an occasion when typhoid fever was the subject for discussion at a meeting of the New York County Medical Association. At this meeting I again called attention to the fact that some cases reported as typhoid were not typhoid, but might probably be a disease due to a paracolon, or as the reader of the evening called it following Schotmüller, a paratyphoid infection.

In conclusion, I wish to say that clinically, as I showed in my paper nearly five years ago, these cases can be readily differentiated from typhoid, notwithstanding they so closely resemble that disease. Under all circumstances I contend that I have the right to claim priority for myself in clinically calling the attention of the medical world to the possibility of some cases like my own being not a typhoid but a paratyphoid infection, and in accurately describing these cases.

I do not believe that my reported cases were of a solitary epidemic, because since then, and almost every summer, I have met identical cases, and at this moment I have two cases in the hospital with a most probable paratyphoid infection.

Dr. Libman: I wish to draw attention to a few points to show that the subject is by no means a simple one. On a previous occasion when this matter was under discussion, Dr. Meltzer threw out the suggestion that in some of the cases referred to there might have existed a mixed infection; cases in which an infection by a paracolon bacillus was followed by an infection by the typhoid bacillus. In connection with this I wish to call attention to the case I am studying in which it is very probable that the reverse occurred. In this case a paracolon ba-

cillus was isolated from the blood twice during life and from the organs post-mortem. Typhoid bacilli could not be found, but the blood of the patient a few days before death gave a positive reaction with the typhoid bacillus in a dilution of 1–250. The blood gave a thread reaction, so-called, with the paracolon bacillus in dilution of 1–20. I would draw attention to the necessity of watching for this reaction at an interval of from four to twenty-four hours after making the test. I think it is highly probable that in these cases the infection with the paracolon bacillus is of a secondary character.

Dr. Ewing: Walker's experiments in Tavel's laboratory would lead one to believe that unless sera are extremely potent and the dilutions very high, they fail to produce the desired results. Dr. Coleman has spoken of the necessity of using different cultures in the differentiation of typhoid species. I have tried for two months to get the Widal reaction in supposed cases of paratyphoid, but have met with no success which would lead me to the routine use of multiple cultures. I believe that the ideas on this subject would be better correlated if we retain the term typhoid in all these cases, and at the same time recognize that the bacterial causes of typhoid fever are more diverse than has heretofore been supposed.

Dr. M. Manges: I would like to comment upon the distinctive character of these cases of paratyphoid, chief among which seem to be the rather abrupt onset and termination, the short cause, the absence of abdominal symptoms, and the failure to respond to the Widal reaction. I believe that a further study of the subject will lead to a simplification and perhaps entire change of some of our views in regard to typhoid. I do not think that these cases of paratyphoid infections can be distinguished clinically from true typhoid, as Dr. Brill seems to think

I regard the differential diagnosis as very difficult, at least in my own experiences.

Dr. Charles Norris: I consider that the term paratyphoid is a very poor one to use, and that in adopting it we are making the same mistake as our forefathers did when they first used the term "typhoid fever". The group of bacilli giving rise to diseases resembling typhoid is a very large one, including many varieties, from the hog cholera bacillus downwards; but it is becoming the custom to recognize nothing as typhoid except that which has the specific bacillus known as the cause of the disease and bearing the same name. Further confusion has been caused by two or three different names having been given to probably the same micro-organism.

Dr. S. J. Meltzer: In some respects I am perhaps responsible for the name "paratyphoid" which has been applied to these cases, and I think that at present it is the most appropriate term that could be used, inasmuch as the clinical symptoms resemble those of typhoid, and one can not very well employ the word "paracolon" because there is no class of diseases known as colon diseases.

Dr. Coleman: I wish to repeat that the reason for calling the cases paratyphoid was that they bore such a strong clinical resemblance to typhoid.

A DEMONSTRATION OF THE TECHNIQUE OF THE DETERMINATION OF THE FREEZING POINT OF BODY FLUIDS BY MEANS OF THE BECKMANN APPARATUS.

F. C. WOOD, M.D.

This apparatus consists of a thermometer with a very finely divided scale and two small glass vessels, one of

which acts as container for the solution to be tested, the other as an air jacket to equalize the action of the freezing mixture in which the vessels and the thermometer are placed. The thermometer can be read to a thousandth of a degree if a lens is used and must be adjusted and tested before use by freezing some distilled water, thus determining the zero of the instrument. While the cooling of the fluid is taking place, constant stirring must be done with a small platinum wire which extends down through one of the corks closing the inner freezing vessel, which contains the fluid to be tested. The whole apparatus is placed in a large jar with a mixture of ice and salt, containing enough salt to reduce the temperature about one to two degrees below zero Centigrade. This will necessitate only a moderate amount of salt, and the mixture should contain a good deal of water. The whole is to be wrapped in a thick felt cover to prevent sudden changes of temperature. As the liquid in the freezing chamber cools the mercury will be noticed to sink gradually below the freezing point of the fluid; and if now rapid stirring be begun and especially if the stirrer be allowed to strike the bottom of the vessel, small particles of ice will be formed and will be whirled up through the rest of the fluid, at which instant the mercury will be observed to rise rapidly for a few tenths of a degree and finally remain constant at a given point, which point cannot be altered by stirring. This is the desired freezing point of the liquid.

The rise of temperature is due to the fact that the fluid has become undercooled; that is, has been cooled below its freezing point. When the ice separates, the latent heat of the water is set free and the temperature

of the mixtures rises in the case of pure water to zero C.; in the case of one of the animal fluids to the freezing point of that fluid. These observations must be repeated three or four times, and the average taken of the readings. They should not vary more than a few thousandths of a degree from each other.

The determination of the freezing point of distilled water, in other words, the zero of the instrument, must be made every day, for when the mercury rises to room temperature point the glass of the bulb takes a set which prevents the mercury returning accurately to zero when it is suddenly cooled. The accurate zero is only reached after the thermometer has been cooled for some time. In making a series of observations, it is well to keep the thermometer in an ice-box, so that the zero will not vary, in which case it will not be necessary to adjust the zero every day.

The under-cooling should be limited to about three or four tenths of a degree, and if, as is frequently the case, the fluid refuses to form ice at this temperature, it is necessary to stir the mixture by raising the stirrer up to a level with the small side tube with which the freezing tubes are equipped, and then to touch this stirrer with a small particle of ice or a frozen portion of the substance which is being tested. This causes a small quantity of ice to form on the platinum ring of the stirrer and when the latter is dropped again into the fluid, formation of ice will immediately begin, and the mercury will rise to the freezing point desired. The apparatus may be simplified somewhat for clinical purposes by using instead of a freezing mixture of salt and ice, a closed vessel containing ether through which is drawn a current of air by means of a small water pump. The rapid bubbling of the air causes evaporation of the water

and a rapid reduction of the temperature of the freezing vessel containing it.

With this apparatus it is often possible to make a determination in ten to fifteen minutes. With the Beckmann apparatus, owing to the large size of the bulb of the thermometer and the necessity for its adjustment, it will usually take about two hours for each determination. Time may be saved by using a thermometer with a fixed zero instead of the Beckmann thermometer, in which case the mercury bulb can be made much smaller; but the results obtained are as a rule not as accurate as those furnished by the original form of the Beckmann apparatus.

The thermometers should be standardized by comparison with some known instrument, or what is better, carry a certificate of the Reichs-Anstalt that they are accurate.

The importance of the determination of the freezing point of animal fluids is that it gives a physical method of determining with very considerable exactness the molecular content of the fluid and with it the osmotic pressure which that fluid exerts. The basis of the whole matter was originally observed by the English physician, Blagden, in 1788. He discovered that the freezing point of a solution was lower than that of a pure solvent and that this lowering of the freezing point was proportional to the amount of substance contained in solution. This observation remained unused until within the last few years, when with the development of physical chemistry, it was found that the molecules of a substance dissolved in a fluid acted just as the molecules of a gas act, and that the osmotic pressure when measured directly was

proportional to the amount of substance dissolved in the fluid, just as the pressure of gas is proportionate to the number of molecules contained in a given volume.

These facts are true without further consideration when the solution is of a certain nature, such as benzol, but when we come to determine the osmotic pressure of an aqueous fluid, we find that the proportion between the quantity of substance present and the pressure is not exactly the same. This has led to the further hypothesis that when substances are dissolved in water they undergo a partial decomposition into ions, and that these ions act as free and independent molecules, thereby raising the osmotic pressure of the fluid proportionally. This occurs only in dilute solutions, such as are found in the human body. Inasmuch as the direct measurement of osmotic pressure is very time-consuming and very difficult, the freezing point method has found general application in the determination of a number of considerations. It has been found, for example, that in cases where the permeability of the kidney is in any way injured—and the best instance of this is seen in a chronic nephritis—that the number of molecules in the blood is increased owing to this inability of the kidney to excrete the molecules into urine. This molecular retention in the blood causes only very slight changes when measured in the ordinary way by specific gravity, but gives a marked increase in the lowering of the freezing point as compared with normal blood. If we take the normal freezing point of the blood as .56° C., we find that the presence of a marked obstruction of the kidney will often raise this point to .71° or .72° C., and thus indicate a retention of molecules in the blood. The method does not give us any clue as to the nature of the substance retained but this may be obtained by chemical analysis. It, however, does give us a measure

of the amount of substance retained and as it is fairly easy to obtain, while chemical analysis requires days for its results, it has been hoped that this method would afford us a rapid and accurate means of diagnosing inefficiency in the excretory power of the kidney. This hope has been partially realized, notably by Koranyi, who has found that not only in experimental nephritis in animals, but in nephritis in human beings, this rise in the molecular content of the blood could be sufficiently demonstrated. Unfortunately for the practical application of this method, there are many complicating circumstances, the chief one being the fact that cyanosis increases the freezing point of the blood, just as does a kidney inefficiency. The method, therefore, does not give us true results when cyanosis is present. A great deal of work has been done in the determination of the freezing point of urine in hope that this might give us a clude to the kidney work, but at present no definite conclusions have been reached. Caspar and Richter have shown, however, that if we collect the secretions of the kidneys separately by catheterization of the ureter, and then make a determination of the freezing point of the urine received from each kidney, we can obtain a proportional value which gives us the relative capacity of each organ. If then we suspect from the reduction of the freezing point in the blood below .56° C., that the kidneys are inefficient, by obtaining the freezing point of the urine from each kidney we can determine the efficiency of each, and in this way discover which kidney is diseased and whether, if one be removed, the other will have sufficient excretory power to handle the products of the bodily metabolism.

An interesting observation of the care necessary to carry out these determinations is shown by work done by Krönig and Fueth, which has recently been published as a criticism of a paper by Veit. The latter obtained different results when he determined the freezing point of the blood of the mother and the new-born child; and the difference was such as to represent a pressure difference between the fœtal blood and that of the mother of five pounds to the square inch. It was difficult to understand how so delicate a partition wall as the chorionic membrane could withstand such a pressure, so Krönig and Fueth undertook a re-examination of the entire subject. After a very careful series of observations they showed that the presence of the blood corpuscles in the fluid to be frozen made no difference in the result. They also determined by a long series of observations, the average error of the thermometer used. This error between two standard instruments was about nine one-thousandths of a degree. Then allowing for this error a series of freezing point determinations were made with the blood from the mother and the child, and they were found to be the same within the limits of error of the instrument.

CONGENITAL STENOSIS OF THE AORTA AT THE ISTHMUS.

E. LIBMAN, M.D.

This specimen is a striking example of a type of congenital cardiac lesion which is compatible with long life. According to Vierordt, there are about one hundred and thirty cases of the malformation on record; it being second in frequency to pulmonary stenosis. The average

duration of life is thirty-four years; one case lived to be ninety-two years old.

The stenosis usually occurs at the isthmus, the ductus remaining more or less open or being closed. The exact site is at the opening of the ductus, just below or above it. The stenosis may be ring-like or funnel-shaped. All degrees of contraction occur down to complete occlusion (fourteen cases of occlusion are recorded).

The collateral circulation is carried on mainly by branches of the subclavian arteries communicating with the intercostal arteries, and with some of the branches of the external iliacs. The development of large anastomosing arteries under the skin of the chest and abdomen is a natural result. The diagnosis can be made in adults rather readily, but hardly in young children, in whom the the large subcutaneous arteries are developed. The diagnostic signs are: hypertrophy of the left ventricle; a systolic murmur heard over any part of the heart, usually loudest at the base or at the apex; the disparity between the pulsations of the arteries of the upper and lower extremities; and the presence of pulsating vessels under the skin of the chest and abdomen in which the wave is from above downwards. This last reservation is made because of the existence of a case published by Türk, in which all the signs of aortic stenosis were found, but the pulsation in the subcutaneous vessels was from below upwards, and the arterial pulsations in the lower extremities were more marked than in the upper. A diagnosis was made of obstruction of the large vessels arising from the arch of the aorta due to specific arterial changes. This was confirmed by the autopsy.

Cyanosis is not a usual symptom in these cases.

The specimen which I have here was obtained at the post-mortem examination of a man, sixty-three years of

age, who was admitted on November 1, 1901, into the service of Dr. Rudisch in the Mount Sinai Hospital. The family history was negative. Previous history: For five years he has suffered from dysponoæ and palpitation. No history of syphilis or gonorrhœa. Marked tabacco habit; moderate alcoholic habit.

Present history: For five months he has suffered from dysponœa, palpitation, œdema of the legs, and diminution of the volume of urine. He has been treated at various hospitals without relief. He now coughs and expectorates much muco-purulent material.

As to the status on admission, I shall give only the main points. The finger tips were cyanostic. There was marked general œdema. *Lungs:* Subcrepitant rales over both; friction rales at the right base. *Heart:* Apex-beat, fifth space, two and one-half finger breadths outside the mammillary line (16 cm. from the median line). Left border, 17 cm. from the median line. Right border, 4 cm. to the right of the median line. Systolic murmur at the apex, not transmitted to the left, and heard very well in the fourth space to the left of the sternum. The murmur was not heard in the vessels of the neck. Pulse, 90. Moderate sclerosis of arteries. Liver and spleen enlarged.

The urine was diminished in quantity. On examination it was clear, acid, specific gravity 1,022; 1% of albumen present; hyaline casts.

The subsequent history was no different from that which is usual in cases of disturbed compensation from other causes. The patient died from an intercurrent erysipelas on February first.

On post-mortem examination there was found marked œdema of the entire body. Under the skin of the anterior thoracic and abdominal walls was a mass of varicose ar-

teries varying in size from $\frac{1}{3}$ to 1 cm. in diameter. The walls of these arteries were thickened and some were completely occluded. In the anterior mediastinum was an accessory thyroid. The heart weighed 730 grams. The wall of the right ventricle measured 1.2 cm. in thickness. The pulmonary artery was notably dilated. The wall of the left ventricle measured 2.4 cm in diameter.

The aorta at its origin measured 8.5 cm. in circumference; at the origin of the large vessels, 14 cm.; at the isthmus, 6 mm. (in diameter); beyond the isthmus, 9½ cm. in circumference; at the lower part of the thoracic aorta, 6 cm. The abdominal aorta at its upper part measured 5 cm. in circumference; at the lower part, 4.8 cm. The right iliac measured 2.9 cm. in circumference; the left about the same. The innominate artery at its origin measured 14 cm. in circumference; the left carotid, 5 cm.; and the left subclavian, 4 cm.

It will thus be noted that there was a considerable stenosis at the isthmus with dilatation in front of the isthmus and below it. The left carotid and subclavian arteries were dilated to twice their normal size. The arch of the aorta at its origin showed but moderate atheroma, as did also the coronary arteries. Directly in front of the stenosis there was a thick spur of calcareous material, and in the dilatation beyond the stenosis the aorta showed very marked atheroma. The remainder of the aorta and the peripheral vessels showed moderate atheromatous changes.

The spleen was much enlarged, weighing 400 grams; and the liver which presented the changes of chronic congestion with some cirrhosis, weighed four pounds.

As a counterpart to this case, I show here a case of stenosis of the isthmus of the aorta from a child one and

a half years old, which died in the service of Dr. Koplik some eighteen months ago.

In this case it will be noted that the isthmus of the aorta is contracted only to about half its normal size, and there were no atheromatous changes present. There was no subcutaneous collateral arterial circulation yet evident.

Discussion.

Dr. Connor: Is dilatation below the stenosis at all common? It seems a strange and inexplicable condition.

Dr. Libman: In these cases various conditions of the aorta below the stenosis had been found. It has either been normal, stenosed, or, as in this instance, dilated. I imagine that the cause of the dilatation was the effort of the blood to get back into the arch of the aorta. It seems probable that little or no blood could be propelled into the thoracic aorta and therefore the collateral circulation through the intercostal arteries might be carrying enough blood into the thoracic aorta to dilate it. To corroborate this point of view I should like to point out that the intercostal arteries which arose from the dilated portion were much larger than those arising from the lower part of the thoracic aorta and from the abdominal aorta. The valvular arrangement on the distal side of the stenosis indicates distinctly that the stenosis was not due to cicatrization of atheromatous ulcers, a condition which one might be expected to exclude in such a case.

A CASE OF ANTHRAX.

L. T. LEWALD, M.D.

The patient, an Italian, fifty-eight years of age, had been employed as a stevedore, and in this capacity had

been engaged in unloading sheep's hides. He was admitted to the Erysipelas Pavilion, Bellevue Hospital, on March 1, 1902. Three days before he had noticed a small pimple in the submaxillary region. On the following day he was seriously ill and unable to go to work. On the third day he was admitted to the hospital. His temperature then was 102°, and it rose rapidly to 108°. Afterwards it dropped to subnormal; but the man continued to grow worse and died on the day following his admission. When he was admitted the only lesion which was noticed was a small ulcerated area about half an inch in diameter with a dark eschar in the center and surrounded by a number of small blebs and a diffuse redness. There was considerable oedema of the face and neck; and this spread rapidly to the chest and also to the forehead to such an extent in the latter direction that it completely closed the right eye. Afterwards signs of cyanosis developed and death occurred about twenty-four hours after the man had been admitted. A smear was made from the wound in the neck a few hours before death, and the examination made at the laboratory confirmed the diagnosis of anthrax. The antopsy was obtained forty-eight hours later and showed the following conditions: The body was well nourished and rigor mortis marked. On the right side of the neck, in the submaxillary region, there was a small ulcer covered by a slight scab. In the surrounding tissue there were a number of small blebs containing a slightly serous fluid. There was a marked swelling and oedema of the face and chest. The brain showed intense congestion. The heart was of normal size. The left ventricle was contracted, the right ventricle dilated. The lungs were congested and the liver showed remains of a few old adhesions. The kidneys showed a slight degree of chronic nephritis. The spleen

and pancreas were normal. The stomach was dilated and the mucous membrane showed signs of chronic gastritis. The epiglottis was markedly oedematous, in fact, sufficiently so to account for death. Smears taken from the left ventricle of the heart showed a considerable number of large bacilli of the anthrax type, and the culture from this source developed anthrax. Cultures from the spleen also showed anthrax bacilli, as did likewise cultures and microscopic examinations of stained sections of the liver. There was very slight parenchymatous degeneration of the liver and of the kidneys on microscopic examination. The lungs and spleen showed acute hyperaemia. The subcutaneous tissue surrounding the area of infection was almost necrotic.

The points of special interest in the case are: The mode of infection, apparently derived directly from the handling of hides; the widespread oedema, starting in the immediate vicinity of the wound of entrance and resulting by direct extension in oedema of the larynx and death; the absence of acute hyperplasia of the spleen or other ordinary evidence of a severe septicaemia, although anthrax bacilli were found in the liver and spleen at the autopsy.

Discussion.

Dr. Conner: In my experience anthrax is much less infrequent in this city than was commonly supposed; and this seems to be just the type of men among whom the disease develops, longshoremen whose duty it is to handle hides. Within the last three or four years, four or five cases have come into the dispensary of the Hudson Street Hospital. Singularly good results have been obtained from early radical excision. As the prognosis depends largely on early recognition of the disease, it is

well to emphasize the fact that cases of the kind are not uncommon and that they are likely to be met with in the dispensary rather than in the hospital ward.

Dr. Ewing: Was there any bacteriological examination made of the blood?

Dr. Lewald: No such examination was made during life?

Dr. Ewing: It is desirable that the blood should be tested with the view of ascertaining whether it was a form of true septicæmia. It is all the more important because to determine the presence or absence of the bacilli in the blood since various conflicting reports which had recently been published as to their presence in the general circulation in cases of human infection.

A CASE OF HODGKIN'S DISEASE.

E. L. DOW, M.D.

This case is one which was observed in the wards of Bellevue Hospital, and which was regarded as a typical case of Hodgkin's disease remarkable for the unusual ulceration of the tonsils.

The history of the patient was as follows: He was a laborer, forty-five years old, had had no disease except pneumonia some eighteen years previous to his admission. No history of syphilis. For the past five years, he had not been as strong as usual nor able to carry out his regular work. The year before his admission to the hospital he noticed a small swelling under the angle of the right jaw. This slowly increased in size, and seven months latter he noticed other hard swellings on the left

side of the neck and in both groins. With the appearance of these groin tumors he also began to notice that he had difficulty in breathing. The tumors were painless. For the past seven months the tumors have been rapidly increasing in size, and are now very large, filling up the entire lateral aspect of the neck, and forming large masses in the groin. Swallowing is difficult and painful. Expectoration has been at times blood-tinged and mucopurulent. He had not lost flesh at the time of his admission to the hospital. When admitted the tumors were found to be circumscribed, irregularly spherical masses quite uniformly firm and elastic without any evidence of suppuration. The skin was freely movable over the tumors. In the neck they were situated immediately under the lower jaw on the floor of the mouth and below the ears, pushing the chin up so that opening of the mouth was limited. In each groin there was a similar character with discrete nodules the size of a large fist. In the axillæ were similar swellings the size of an English walnut. Indistinct masses could be palpated through the abdominal wall in the iliac and lumbar regions. The patient appeared fairly well nourished but anæmic. The dysponoea was extreme and the breathing stridulous. The jaws could not be separated sufficiently for a pharyngeal examination. The heart, lungs, liver, and spleen showed no marked abnormalities. On admission his temperature was 101.6°; pulse, 120; respiration, 30.

During his stay in the hospital he had an irregular temperature varying from 100° to 103°. Examination of his blood after admission revealed 3,600,000 red cells and 2,900 whites to the ccm. The number of leukocytes remained low until his death. The urine was normal. Sputum examination showed no tubercle bacilli. During his stay in the hospital, the dysponœa and difficulty in

swallowing became so great that he took no food voluntarily. Death due to pulmonary œdema.

The autopsy, six hours after death, showed a fairly well nourished body, slight œdema of the legs. In the cervical, inguinal, iliac, lumbar, and mesenteric regions were found the tumors previously mentioned. The secondary and tertiary lymph nodes were not involved. Those in inguinal regions each weighed 10 oz. They were composed of numerous lymph nodes enlarged and bound tightly together. Everywhere the nodes were discrete. In the iliac chain they showed little fibrous structure. No evidence of suppuration existed, though some of the nodes broke down under pressure. The anterior mediastium contained a few large lymph nodes; the posterior contained many nodes the size of English walnuts which extended round the roots of each lung, forming masses the size of a fist. Bronchi, trachea, and larynx were intensely congested. The tonsils were deeply ulcerated and hyperplastic. The entire pharyngeal lymphoid tissue was markedly hyperplastic. Chronic changes were found in the kidney. The intestines showed hyperplasia of Peyer's patches and solitary lymph nodes. One Peyser's patch projected a centimetre into the lumen of the intestine, looking very much like a small mushroom. The surface was ulcerated and there was a post-mortem invagination of this portion of the intestine.

Microscopic examination of the ulcerated tonsils and lymph nodes revealed only round cell growth with hyperplasia in all but the iliac, lumbar and mesenteric nodes. There was no evidence of tuberculosis.

The deep ulceration of the tonsils apparently occurs more frequently in those cases running an acute course. A number of observers have noted such a condition in connection with acute inflammations of the throat.

Discussion.

Dr. Wood: In some of the recent text-books it is stated that the lymphocytes in Hodgkin's disease are relatively increased while in cases of tuberculosis simulating Hodgkin's disease no such increase is present. In the cases of Hodgkin's disease, however, which I have been able to examine no such relative lymphocytosis was found. I wish to know if Dr. Dow has noticed this point in his case.

Dr. Dow: I have not.

Dr. Field: In view of the recent discoveries in regard to the pathology of Hodgkin's disease it is important that all such cases should be put through a careful examination for tubercle bacilli.

Dr. Ewing: I would like to add not only for the tubercle bacillus but for other bacteria as well.

Dr. Dow: There was no evidence of tuberle bacilli in the tonsils nor in the lymph nodes. I would like some expression of opinion from the members of the Society as to whether they consider the ulceration of the tonsils a primary or secondary condition. If secondary, why should it occur in the tissues of the tonsils rather than in the hyperplastic lymph nodes?

DEMONSTRATION OF HAEMOLYSIS.

F. C. WOOD, M.D.

The specimen to which I wish to call your attention are those which illustrate the action of foreign sera on corpuscles of different animals. I have selected rabbit's blood and dog's serum to show their mutual interactions.

If we add to rabbit's red blood corpuscles, suspended in physiological salt solution, a small amount of dog

serum, the hæmoglobin of the rabbit corpuscles will be extracted and dissolved in the fluid. The same is true if use bullock's serum and rabbit's corpusles. This reaction may be prevented by heating the serum to 58°C., for half an hour. This heated serum will then not cause any laking of the corpuscles. The laking action may be restored by the addition of fresh serum from the same animal from which the corpuscles were removed. For instance, if we treat rabbit's corpuscles with bullock's serum which has been heated there will be no reaction. If, however, we add a small quantity of fresh rabbit serum to the mixture, the hæmoglobin will be extracted. This is evidently not due to any action of the rabbit serum, for such serum is isotonic to its own corpuscles, but is due to the addition of a certain complementary body which enables the altered bullock serum to again lake the red cells by a process which is not as yet fully understood.

CARCINOMA OF THE PANCREAS.

A. E. THAYER, M.D.

The clinical history of this case dates from five weeks before the patient was admitted to the hospital. At that time he began to have severe pains in the lumbar region which radiated forward into the abdomen, were partly relieved by pressure, and which lasted half a day. Such attacks occured at intervals until admission. He vomited at times, but there was no blood in the vomit. His strength and flesh had steadily diminished and for about three days he had been jaundiced. His feces had been formed and yellow.

On admission the patient was poorly nourished; respiration was wholly thoracic; the abdomen was tense,

retracted, and generally tender on palpation; and in the epigastrium and reaching into each hypochondrium there was a prominent mass of irregular outline. Neither liver nor spleen could be made out. The urine contained no albumin nor sugar, but was deeply bile-tinged. The entire surface was deeply jaundiced.

The patient's temperature varied between 99° and 101°; respiration about 28 to 30, and pulse 120 to 130, while he was in the hospital. An exploratory operation was performed, and from that time he sank, having diarrhœa, and vomiting a dark fluid till he died from exhaustion about three weeks after admission.

At the autopsy there was found a large mass in the pancreas, the older portions about midway of its length showing cystic softening and involving the spleen and the left adrenal. The lymph nodes about the gland were generally involved. On the upper posterior aspect of the liver there was a single direct metastasis, which had nearly penetrated a large branch of the hepatic vein. The colon contained bluish-gray feces; the gall-bladder was a little distended. There was no dilatation of the stomach nor contraction of the duodenum. On microscopic examination the tumor was found to be adenocarcinoma. No trace of pancreatic tissue was left, and almost no splenic.

I wish also to present a case of suppurative cholangitis, due to biliary calculi impacted in the common duct. In this case there is no clinical history. The patient, a Russian of about forty-five years, was intensely jaundiced and much emaciated. Post-mortem six gall-stones were found impacted in the common bile duct, lying in a collection of pus. All the larger branches of the bile ducts through the liver contained pus and small grains of black pigment; and in the left lobe there were several abscesses,

about 3 to 5 cm. in diameter. The gall-bladder was much distended, contained grayish-green, thickened mucous fluid and grains of pigment, and from its inner wall there grew a small soft fibroma. There was no distension of the pancreatic duct, no interstitial pancreatitis nor fat necrosis. There was complete hyperplasia of the left kidney.

The two cases are of interest as showing how little reliance can be placed in Courvoiser's law, so-called: that with bile stones the gall-bladder is small, and with malignant disease of the common duct it is large. A number of recent cases demonstrate that this view is untrustworthy. The relation between occlusion of the common bile duct and pancreatitis and fat necrosis also appears to be slight from these cases. Further, in each there might have been sugar in the urine, especially in the first case where the pancreatic tissue was wholly replaced by a neoplasm, but, so far as the records show, there was none.

CARCINOMATOUS ULCERATION OF DUODENUM, WITH ABCESSES OF LIVER AND PANCREAS.

JOHN H. LARKIN, M.D.

For several years the Society has been receiving reports on perforating ulcers of the duodenum, but the present case is of exceptional interest because of the complications disclosed by the specimen, although I must apologize for the incompleteness of the history of the case.

The patient was a woman, forty-six years of age, an Icelander, and it was difficult to obtain any facts beyond

this, that for about two months she had been ill, simply with pains in the lumbar region. No physician had seen her until two weeks previous to her being admitted to the hospital. At the time of her admission the case was put down as one of ordinary destitution and starvation. Three hours before death, she had a large and copious bloody movement from the bowels, then went into a collapse and died.

At the autopsy an interesting condition of the organs was revealed; but before speaking of this I wish to say a word about the symptoms. At no time, so far as could be ascertained, had there been any abdominal pain or vomiting. At the autopsy I found the body markedly emaciated. There were no evident signs of peritonitis; some clear serum in the abdominal cavity. All the viscera were normal except that they were pale and blanched from loss of blood. The sudden hemorrhage and collapse pointed to an intestinal lesion. I expected to find an abdominal aneurysm with perforation into intestine, but in this I was mistaken. In the stomach there were two pints of clooted blood, but the mucous membrane was perfectly normal. On going down to the duodenum I discovered a large, irregular ulcer, about the size of a dollar, with several small nodules at the periphery. In the center was a large necrotic mass directly through the duodenum, and the adjoining liver tissue was extremely necrotic, making a large abscess cavity filled with pus and recently coagulated blood. In the middle of pancreas was a small metastatic nodule, firm and fibrous. The blood vessels were dissected up and the superior mesenteric vein was found necrotic and leading directly into the abscess cavity. Microscopical examination of sections taken from edge of ulcer in duodenum and mass in pancreas showed adenocarcinoma.

MULTIPLE EMBOLI OF MESENTERIC ARTERIES.

C. PHILLIPS, M.D.

The case is that of a physician, thirty-three years of age. His past history showed that he had always been a well man and had never veneral disease or gout. He gave a vague history of attacks of pain, five years before, in the right inguinal region, lasting two or three days.

The patient was quite alcoholic, drinking beer and whiskey, and an immoderate eater. For three or four nights before the onset of present trouble, the patient had been intoxicated. He awoke in the morning about four o'clock, complaining of severe abdominal pain which was constant and general. He rapidly developed tympanites and the entire abdomen became generally painful on palpation. There was no localize tumefaction or increased resistance to pressure. He had considerable hiccough and vomited a few times, but no blood. It was impossible to get a movement of his bowels by cathartics or high rectal enema. Pulse rate was between 80 and 90 and he had no fever. During the first twenty-four hours, he passed but 8 oz. of bloody urine.

About forty-eight hours after the onset of the symptoms, he was operated upon by Drs. Bull and Coley. The abdominal cavity contained about 1500 c.c. of serosanguineous fluid with some flakes of fibrin. The small intestine was much cyanosed, markedly distended with gas, and here and there were small hemorrhages under the peritoneum. The omentum was much swollen and contained also many hemorrhagic food. There was no obstruction of the intestine found. The patient died four hours after the operation, and an autopsy was per-

formed by Dr. Harlow Brooks, to whom I am indebted for the following notes:

On opening the peritoneal cavity about 300 c.c. of creamy pus escapes, and the parietal and visceral layer of the peritoneum is covered with a thin layer of fibrinous pus. The entire gut is distended with gas, particularly the cæcum. The peritoneal capillaries are much injected both on the walls and over the intestines. The omentum is very short and thick and laterally studded with small spherical injected gangrenous nodules, which appear not unlike Delaware grapes. Evidently they represent minute embolisms of the omental vessels. The omentum is much walled together, as from previous disease. The mucosa of the stomach shows evidence of chronic gastritis and numerous old healed ulcerations of small size. The appendix is sharply folded on itself and surrounded by œdematus adipose tissue and old adhesions. It is about 2 cm. long, and has evidently ruptured with complete loss of the distal two-thirds. There are no evidences of recent inflammation.

The mucosa of the small intestine as a whole is much congested but in places is chæmic. There are a few minute emboli of the submucous vessels but more frequent and larger lesions of the same character on the peritoneal surface, resulting in small gangrenous patches.

The mesentery of the lower folds of the ileum shows a very marked congestion with the presence in the small vessels of minute emboli. This condition becomes more frequent in the upper folds of the mesentery where the emboli are larger. The mesenteric vessels as a whole show the most marked evidence of recent endarteritis; while here and there are portions of the intima which are calcified.

The pancreas is riddled with fat infiltration. The

calibre of the artery is about that of the axillary with enormously thickened intima. The pancreatic duct is gangrenous in part, apparently from thrombosis of its nutriment artery. There is extensive infiltration of the pancreatic interstitium, apparently inflammatory. The liver is moderately fatty and considerably congested. The kidneys show evidences of acute nephritis with very evident fatty degeneration.

The heart is large and the muscle shows extensive fatty degeneration. The valves are thin but competent. In the right auricle is a preformed clot, evidently of two or three days duration. It is distinctly laminated, very firm, and on the surface it is covered with small granulomatous masses. It is firmly adherent to the superior external wall of the auricle and shows beginning organization. The coronary muscles are much thickened and atheromatous. The aorta is of small calibre but atheromatous throughout. The lungs are congested and show here and there small recent hæmorrhagic infarctions.

Cause of death: Multiple infarction of the pulmonary and mesenteric vessels from thrombosis of the right auricle; this condition occurring in acute alcoholic toxæmia.

No cultures were taken.

ENDOTHELIOMA OF SEROUS CAVITIES.

C. W. FIELD, M.D.

The patient, from whom the specimens were taken, was a woman fifty-six years of age, a widow with three

children. The history of the case was negative, except that for some time she had a hacking cough and that her stomach had always been weak. She had been losing strength for two years. Pulmonary symptoms afterwards developed, but there was at this time no vomiting or gastric disturbance. The woman was admitted to the Presbyterian Hospital on December 20, 1901, after having been affected by nausea and diarrhoea for three weeks. A physical examination made on January 27th showed a marked adhesion over the whole of the right lung. The patient continued to grow worse and died soon afterwards, having previously vomited a large quantity of bloody serum.

At the autopsy a very thorough examination was made of all the organs and it was found that the pleura was very much thickened, apparently by a tuberculous lesion. On sectioning the thickened mass, however, it was found to be a new growth of alveolar type, probably to be classed with the primary endotheliomata of the serous cavities. There was also found a tumor involving the optic nerves which was of the same morphological type. The only indication of this new growth from a clinical point of view had been headache of which she complained while in the hospital. The exact site of the origin of the new growth was therefore somewhat difficult to assertain, but it seemed probable that the pleural tumor was primary and that of the optic nerve secondary, though the existence of two tumors developing simultaneously was not impossible.

Discussion.

Dr. John H. Larkin: From the microscopical examination it seems to me that the tumor may have been primary in the optic nerve.

COMPLETE DOUBLE URETER ON EACH SIDE, PERVIOUS THROUGHOUT.

T. C. JANEWAY, M.D.

In presenting this specimen of complete double ureter on each side, I need not detain the Society with any lengthy description, as it is simply a very rare anatomical abnormality. In looking over the transactions of the London Pathological Society and Virchow's Archiv, I have found only one exactly similar case, reported by Ewart. Specimens showing ureter double for a portion of its length on one side, and on the other side one normal ureter and one abnormal one emptying into neck of bladder or prostatic urethra, have been encountered by Weigert, Hoffmann, Tangl, and Wood. Double ureter on one side only seems to be a much more more common condition, cases having been reported by Weigert (seven in one year), Thompson, Durham, Kelly, and Voelcher.

This specimen was taken from a middle-aged woman, who died of chronic diffuse nephritis. It shows the separation of the pelvis of the kidney into two distinct cavities, common to all previous cases, but is unusual in that there is no crossing of the ureters, and that the upper ureter in each side opens above the lower in the bladder, the opposite of Wiegert's findings.

TWO CASES OF LYMPHOCYTOSIS SIMULATING LEUKAEMIA.

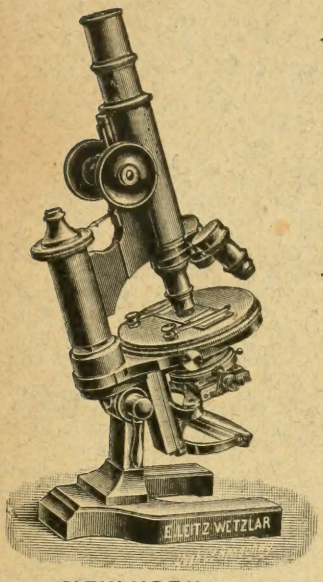
FRANCIS C. WOOD, M.D.

The first case was that of a child with a tuberculous lesion of the hip, the lymphocytosis disappearing after drainage of the abscess; the second case was a fatal case

of anaemia without lesions in the bone-marrow. -Studies of the blood showed marked lymphocytosis in both cases.

Discussion.

Dr. Ewing: These cases are important additions to the scanty knowledge which exists on this subject and shows the importance of examining other things besides the blood before accepting a diagnosis of lymphatic leukaemia. One feature of extreme interest in the second case is its resemblance to pernicious anaemia. At the same time it differs so much from reported cases of pernicious anaemia without bone-marrow changes, that I would hesitate to accept it as such without study of all the antecedents.



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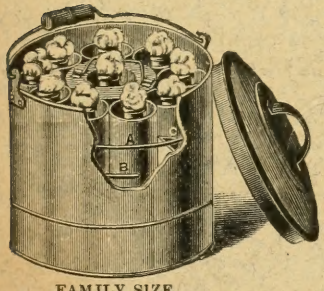

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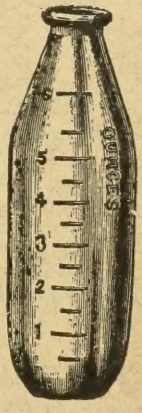

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